Exterior Surface Dust Lead, Interior House Dust Lead and Childhood Lead Exposure in an Urban Environment

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ABSTRACT

The impact of urban lead exposure is being examined in a prospective study of several hundred children followed from birth to five years of age. A wide range of social, behavioral, biological and environmental factors are being assessed at approximately one year intervals beginning at birth. Previous analyses on this cohort have indicated a strong relationship between hand lead and hand-to-mouth activity and suggests that this is an important mechanism of inadvertent ingestion of lead in infants and young children. The present analysis was undertaken to examine the joint influence of lead in exterior surface dust and interior lead-containing painted surfaces on lead levels in house dust. In addition the joint influence of exterior and interior surface dust lead on children's hand lead content and blood lead concentration was examined. At 18 months of age 38% of the observed variation in blood lead was accounted for by hand lead and dust lead. Interior paint lead and exterior surface dust lead accounted for 52% of the observed variation in interior surface dust lead concentration. Exterior surface dust lead, obtained from exterior surface scrapings, indirectly influenced blood lead through its impact on interior house dust lead and children's hand lead content, but had no observable direct impact on blood lead.

INTRODUCTION

Deliberate ingestion of soil by children is a recognized mechanism through which some young children might incur lead poisoning (2). A more frequently occurring mechanism is thought to be the inadvertent ingestion of lead containing dust (cf, 6,7,9). This inadvertent ingestion is hypothesized to occur as a result of mouthing of hands, toys and food which have become contaminated with lead-containing surface dust. Mouthing is a developmentally normal behavior in infants between 3 and 18 months of age. Since very young infants spend a considerable amount of time crawling or playing on the floor, mouthing of hands and toys is a plausible mechanism for the inadvertent ingestion of lead contaminated house dust. Lead in interior house dust might originate from lead-containing paint or from sources exterior to the residence, e.g. exterior surface dust, soil and exterior lead-containing paint. While many qualitative models have been proposed to describe the pathways by which environmental lead exposure occurs during childhood (1,6,7) few studies, if any have attempted to provide quantitative estimates of the relative contribution of the numerous hypothesized paths of exposure.

The Cincinnati Lead Study, which is a prospective study of the mechanism of childhood lead exposure and the effects of lead exposure on deve-

lopment was initiated in 1980 (4). It provides an opportunity to develop quantitative exposure models based on a large urban cohort. Objectives of this interim report are to: a) describe patterns of lead exposure in and around houses occupied by an inner city cohort of young children, b) test the hypothesis that in normally developing children there is a demonstrable and statistically significant path of lead contamination, extending from lead in exterior surface scrapings (PbSS) to lead in interior house dust (PbD), which in turn contaminates the hands (PbH) of children resulting in blood lead (PbB) elevations.

EXPERIMENTAL MATERIALS AND METHODS

Subjects. Children in this study were drawn from a predominantly low socioeconomic, black, inner city neighborhood. Children were entered into the study at birth. Progress in social, behavioral and cognitive development was measured and medical evaluations were made at regular quarterly intervals throughout the study. A more complete description of the full study cohort and study design has been reported elsewhere (4).

The sample in this analysis was restricted to 81 children, 18 months of age with complete data with respect to measures of PbB, PbD, PbH, PbSS and qualitative evaluation of the exterior of the dwelling unit.

Blood Lead Determinations. Blood samples were obtained by venipuncture. All blood samples were analyzed for lead by anodic stripping voltammetry (ASV) with the use of an ESA Model 3010A instrument. Details of the analytical procedure and proficiency have been reported previously (4,11). Prior to data analysis, the PbB values were transformed to their natural logarithm to normalize the statistical distributions.

Qualitative Housing Evaluation. The exterior of all dwellings occupied by study participants were evaluated with respect to age, condition and type of dwelling. A more detailed description of methods of evaluation has been reported (8). Only those houses which could be represented by one of the four most frequently observed housing categories were used in this analysis. These are Public Housing (25%), Rehabilitated Housing (36%), 19th Century Satisfactory (11%), and Deteriorated or Dilapidated Housing (27%), with the percentages of cohort children at 18 months of age residing in each category shown parenthetically. Preliminary analyses have demonstrated a strong relationship between housing categories, interior Pb levels and blood Pb levels (5,8). This strong association is largely due to the fact that Pb-based paint was commonly used in housing built prior to WWII. The older inner-city housing stock in which many of the study subjects reside, remains inadequately rehabilitated and in a chronic state of disrepair.

Environmental Lead Determinations. Environmental samples were collected in the residence of each study participant when they were between 7 and 19 months of age. If the child's family moved during this interval, an additional assessment was performed in the new residence. Three types of environmental Pb measures were statistically analyzed in this report: interior surface dust Pb (PbD in ppm and ug/m^2), hand Pb (PbH in ug) and interior paint Pb hazard (XRFHAZ). Interior surface dust was collected by three sweeps of a measured area using a 2-L/min vacuum pump. Dust samples were collected in five different locations within the interior of the residence unit.

Exterior surface dust scrapings (PbSS) were collected by scraping the exterior surfaces near the dwelling with a stainless steel spatula and placing the sample in a plastic bag. The scraped surfaces were either paved with asphalt, concrete or brick or were composed of hard-packed soil

devoid of vegetation. Samples were collected from areas in which the subject played and/or from immediately outside the dwelling unit entry. Surface scrapings, rather than soil cores are being reported since it is the surface lead content which is more accessible to the child and more likely to be transported into the residence. Hand lead (PbH) samples were obtained from the surface of the child's hands by three repeated wipings of each hand with a total of six wet wipes. Details of sample collection and analysis have been described elsewhere (10).

Recovery studies for both interior surface dust Pb and hand lead sampling procedures have demonstrated a high mean recovery rate (84%). Environmental samples and blanks are predigested, evaporated and evaluated for lead using atomic absorption (10).

Paint lead was evaluated using x-ray fluorescence (XRF) on a maximum of 15 painted surfaces within the dwelling. For each XRF reading, the environmental technician also rated the primary (predominant fault) and (if appropriate) the secondary condition of the painted surface. These values vary from 0 to 10, where high values indicate poorer surface quality. A paint hazard score (XRFHAZ) for each residence was derived from a linear combination of the product of the XRF measurements and the condition code values for the painted surface. This produces a weighted average score which takes into account not only the Pb content of the painted surface but also the (potential) availability of Pb which migrates from the painted surface in the form of dust and paint chips to children. All environmental measures (PbD, PbH, XRFHAZ, and PbSS) were transformed to their natural logarithm to normalize the statistical distributions.

Data Analyses. Descriptive statistics include geometric mean (GM) and geometric standard deviations (GSD) for each type of environmental sample. Inferential statistics include Pearson r correlations between log transformed environmental lead and blood lead measures. Structural equations analyses (3,5) were undertaken to examine the intercorrelation and relative contribution of various sources of exposure and routes of exposure to observed blood lead concentration. The latter technique permits the estimation of and testing for the presence of a hypothesized set of direct and indirect influences on an outcome, e.g. PbB, in a complex set of intercorrelated data (3,13). The SAS SYSREG computer program (12) was used to estimate the structural equations.

RESULTS AND DISCUSSION

Levels of lead exposure in this inner city cohort were strongly influenced by the type of housing being examined (see Table I). Even though these dwellings are located in a confined area of the inner city (<five sq. mi), there is a very large range of housing type and associated lead content. For example, PbD ranged from 82 to 13,820 ppm and PbSS ranged from 76 to 54,519 ppm across the four different house types in the study area. However, certain patterns of exposure are observable. Lowest levels of PbD and XRFHAZ are observed in public and rehabilitated housing since these dwellings are newer or recently renovated and better maintained. Levels of PbD are very high within 19th century satisfactory and deteriorated dwellings. PbSS is quite low outside public housing units, while being considerably higher and more variable outside other house types. Seventy-five percent of the residences occupied by 18 month olds had PbSS or PbD levels >1000 ppm. In 72% of the residences, exterior lead concentrations (PbSS) were greater than interior concentrations (PbD).

The observed variability of lead loading on interior surfaces (ug/m^2) was much greater than the observed variability of lead concentrations (ppm)

TABLE 1. GEOMETRIC MEANS (GM) AND, GEOMETRIC STANDARD DEVIATIONS (GSD) AND RANGES FOR PbB AT 18 MONTHS AND ENVIRONMENTAL DATA, BY HOUSING TYPE

		PbB(ug/dl)			PbH(ug)			PbD(ppm)		
Housing Type	n	GM	GSD	Range	GM	GSD	Range	GM	GSD	Range
Public	20	12.84	1.46	6.5-25.5	6.80	2.17	1-33	435.87	1.80	139-1231
Rehabilitated	29	15.08	1.42	6.0-32.5	5.89	2.32	1-28	566.15	2.11	82-1924
Satisfactory	9	14.32	1.44	9.0-27.0	5.91	4.08	1-60	1,847.29	2.71	485-7598
Deteriorated	22	26.03	1.57	13.5-53.5	14.74	3.74	1-191	2,476.63	2.94	393-13820
All Houses	80	16.91	1.61	6.0-53.5	7.80	2.99	1-191	899.85	3.05	82-13820

		PbD(ug/m^2)			XRF hazard			PbSS(ppm)		
Housing Type	n	GM	GSD	Range	GM	GSD	Range	GM	GSD	Range
Public	20	380	2.38	80-1840	.88	2.28	.23-5.76	247.88	1.96	76-812
Rehabilitated	29	470	3.15	40-4320	.48	2.32	.04-2.49	1654.49	2.56	253-11889
Satisfactory	9	1160	4.56	170-8000	5.79	5.13	.10-31.92	7361.54	3.81	1500-54519
Deteriorated	22	3160	6.39	80-170000	11.94	2.51	1.42-68.54	2791.19	3.91	108-25180
All Houses	80	810	5.05	40-70000	1.74	5.41	.04-68.54	1360.32	4.67	76-54519

in these residences. Because of this, a loading estimate obtained from only one area of the residence or from a small surface area was not representative of loadings on other surfaces within the residence. Lead loading on floor samples tended to be much higher in corners and along walls.

As noted in previous reports (8) the hazard to lead based paint in poor condition was greatest in deteriorated 19th century dwellings. Although some high lead paint was detected in public and recently rehabilitated dwellings, the condition of the painted surface was usually good and thus posed no immediate hazard to the children

The pattern of PbH levels and PbB concentrations were less consistent across house types than PbD and PbSS. However, this is to be expected since 18-month olds are capable of exploring a wide range of areas within and around the dwelling. Furthermore, PbB at 18 months, in part, reflects earlier exposures. Since this cohort moves frequently, current PbB is in part due to exposures incurred in previous residences. The PbB concentrations observed in this cohort indicate that a significant fraction of these 18 month olds were subjected to undue lead exposure.

TABLE II. INTERCORRELATIONS AMONG ENVIRONMENTAL AND BLOOD LEAD DATA OBTAINED FROM 18-MONTH OLDS

	Ln(PbB) ug	Ln(PbH) ppm	Ln(PbD) ppm	Ln(PbD) ug/cm^2	Ln(XRF/ Hazard)
Ln(PbH) ug	.52				
Ln(PbD) ppm	.53	.47			
Ln(PbD) ug/m^2	.39	.48	.80		
Ln(XRF/ Hazard)	.44	.31	.64	.54	
Ln(PbSS) ppm	.30	.20	.57	.41	.41

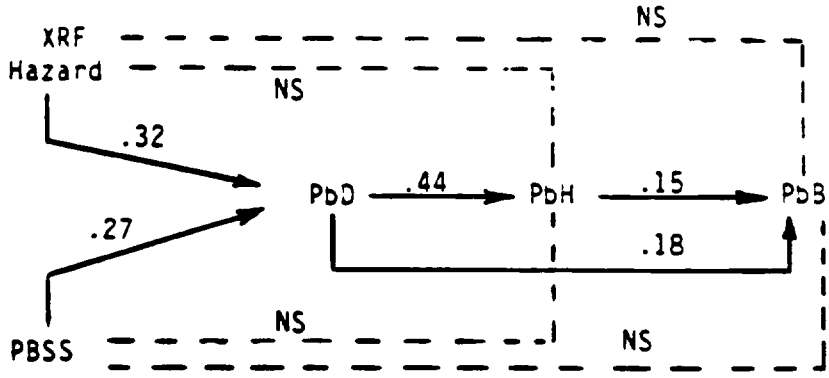
The intercorrelation among the measures is shown in Table II. All reported correlations are significant at $p<.001$. PbB was highly correlated with PbH and PbD as noted in previous reports (5). Lead in surface dust expressed as a concentration (ppm) was very highly correlated ($r=.80$) with amount of lead per unit area (ug/m^2). Because of this high correlation, either expression of PbD correlates well with PbH and PbB. This is contrary to other reports that find the ug/m^2 measure to be more highly correlated with PbB and PbH (6). The explanation is likely to be that, in the Cincinnati study area, those dwellings which contain the highest concentrations of Pb in dust are deteriorating most rapidly and have the poorest maintenance, resulting in both high concentrations and loadings. The correlation between interior paint (XRFHAZ) and exterior PbSS ($r=.41$) is at first somewhat surprising. However it probably reflects the strong contribution of exterior paint to levels of lead found in exterior surface scrapings. Years of building deterioration and renovation (stripping of exterior paint or sand blasting of painted surfaces) have resulted in the

contamination of large areas of the inner city.

Table III summarizes surface dust lead concentration (ppm) and loading (ug/m^2) by house type and location of the sample within the residence. PbD concentrations at the entry and windowsills were considerably higher than PbD samples obtained from floor areas in the interior of the residence. This gradient of highest levels at the outer edge of the residence was seen across all house types. Lead loadings (ug/m^2) were highly variable with the greatest variability seen in samples obtained from windowsills.

The direction of the lead gradient within the home, coupled with a gradient of higher lead concentrations in PbSS than PbD suggest that there is a movement of lead from the outside to the inside of the residence. If this is true for most inner-city dwellings, then any efforts at interior paint lead abatement must be coupled with an effort to reduce exterior PbSS. Otherwise, a "lead-paint abated" residence will soon become recontaminated with lead. In order to estimate the contribution of PbSS to interior PbD and PbB in the presence of interior sources of lead paint (XRFHAZ), a structural equations analysis was undertaken.

Figure 1 shows the structural equations model and equations obtained on this data set. Solid lines indicate that a statistically significant coefficient was found between the two variables, after controlling for all other variables in the model. Dashed lines indicate that no significant coefficient was found for that pathway. Arrows indicate the presumed direction of a causal influence. Numbers adjacent to the lines are parameters estimates. Results of this analysis indicate that XRFHAZ and PbSS contribute to PbD which in turn contributes to PbH. PbSS was found to have no direct effect on PbH or PbB. However it clearly has an indirect effect mediated through interior surface dust. PbSS and XRFHAZ accounted for 52% of the variance in PbD. The lack of a path from XRFHAZ to PbH and PbB is not surprising since this would imply that paint chips were adhering to the hand or being deliberately ingested, both of which are low probability events in the study population. Rather the results support the hypothesis that peeling paint is eventually ground into dust which then contaminates hands, toys and food.



Structural Equations: R^2

Ln(PbB) = 1.276 + .152 Ln (PbH) + .182 Ln (PbD) .38
Ln(PbH) = -0.966 + .444 Ln (PbD) .22
Ln(PbD) = 4.691 + .325 Ln (XRFHAZ) + .268 Ln (PbSS) .52

All coefficients are significant at $p<.05$; NS=Not Significant

FIGURE 1 - REDUCED STRUCTURAL MODEL FOR 18 MONTH OLDS

TABLE III. GEOMETRIC MEAN SURFACE DUST LEAD CONCENTRATION (ppm) AND LOADING (ug/m^2) BY HOUSE TYPE AND LOCATION WITHIN THE HOUSE

		20th Century Public	19th Century Rehabilitated	19th Century Satisfactory	19th Century Deteriorated	All Houses
Interior	ppm	361	498	1422	1541	699
Surface	ug/m^2	290	310	850	1380	500
Dust Lead	n	20	29	9	22	80
Window	ppm	608	889	--	14186	2080
Sill	ug/m^2	990	890	--	13770	1760
Dust Lead	n	11	16	3	12	43
Entry	ppm	572	804	2540	2670	1097
Area	ug/m^2	590	1000	3040	3330	1290
Dust Lead	n	14	18	7	13	53

--Not reported, insufficient sample size

The lack of a direct path from PbSS to PbH and/or PbB suggests several possibilities. It might be that these 18 month old children are not coming in direct contact with exterior surface dust. Given the age of the children and the fact that samples were collected throughout the year, such an explanation seems unlikely. An alternative hypothesis is that the lead-containing fraction of PbSS, in large part, consists of particles too big to adhere to the hand. That fraction of particles which do adhere might be more similar in size to that found in house dust. Models developed with other age cohorts and characterization of PbSS particle size distribution will clarify these issues. The impact of PbSS on PbD after controlling for XRFHAZ is shown in Figure 2. The x-axis extends from 0 to 10,000 pm Pb which encompasses 91% of all observed PbSS concentrations. Increasing PbSS from 0 to 1000 ppm results in an expected increase in PbD from 130 ppm to 830 ppm.

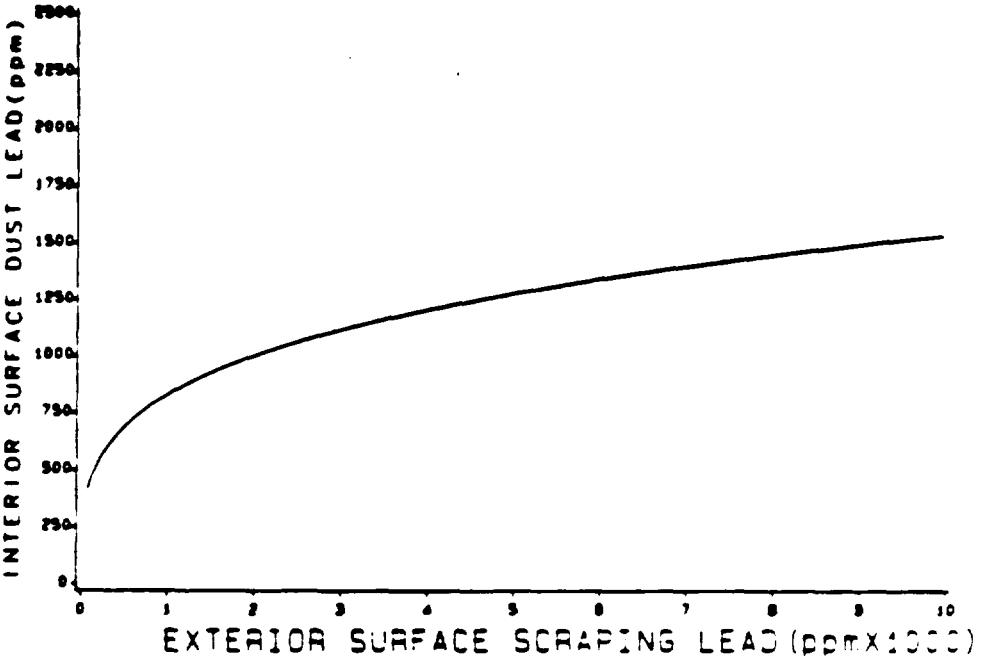


FIGURE 2- PREDICTED LEAD CONCENTRATIONS IN DUST (PbD) FOR GIVEN LEAD CONCENTRATIONS IN PbSS. THIS RELATIONSHIP REFLECTS ADJUSTMENT (CONTROL) FOR THE CONTRIBUTION OF XRFHAZ. THE REDUCED (ADJUSTED) STRUCTURAL EQUATION IS: ln (PbD)=4.871 + 0.268 ln (PbSS).

Figure 3 depicts the expected indirect effect of PbSS on PbH after controlling for XRFHAZ and PbD. Increasing PbSS from 0 to 1000 ppm results in an expected increase in PbH from 3.3 ug per two hands to 7.5 ug.

Figure 4 shows the expected increment in the blood lead levels of 18 month olds. A 1000 ppm increase in PbSS, from 0 to 1000 ppm, results in an indirectly mediated increase in PbB from 10.4 ug/dl to 16.6 ug/dl after controlling for XRFHAZ and the mediating influence of PbD and PbH. As expected given the log-linear relationship between Pb exposure and PbB, the increment in PbB is less, 0.76 ug/dl, for the next 1000 pm increment in PbSS, i.e. from 1000 ppm to 2000 ppm.

Future refinements of this model will incorporate social, behavioral

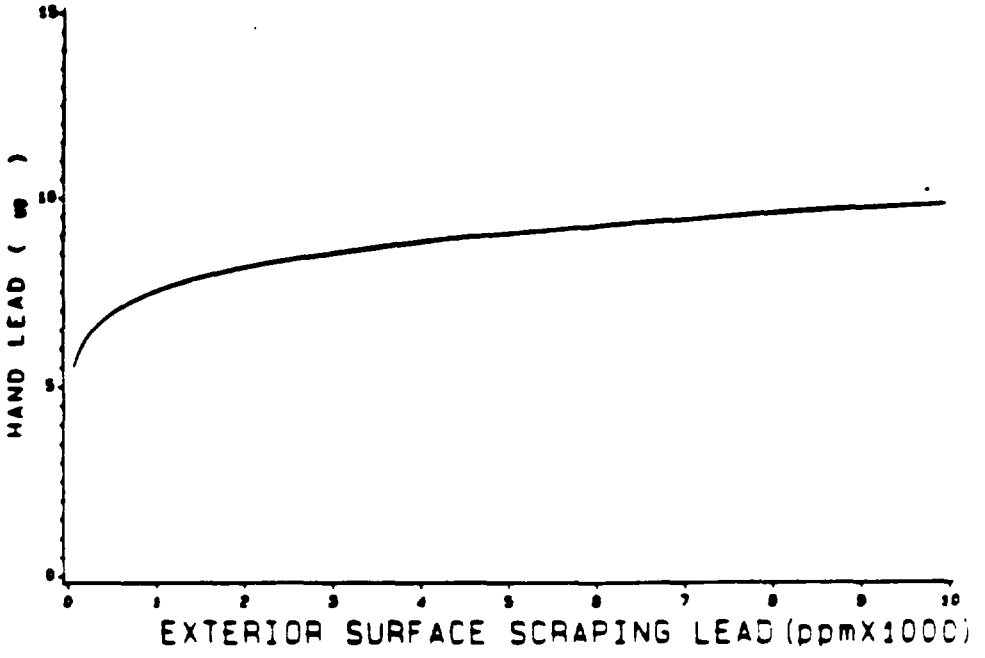


FIGURE 3- PREDICTED LEAD LEVELS ON HAND (PbH) FOR GIVEN LEAD CONCENTRATIONS IN PbSS. THIS RELATIONSHIP REFLECTS ADJUSTMENT (CONTROL) FOR XRFHAZ AND PbD. THE REDUCED (ADJUSTED) STRUCTURAL EQUATION IS: ln (PbH)=1.196 + .119 ln (PbSS).

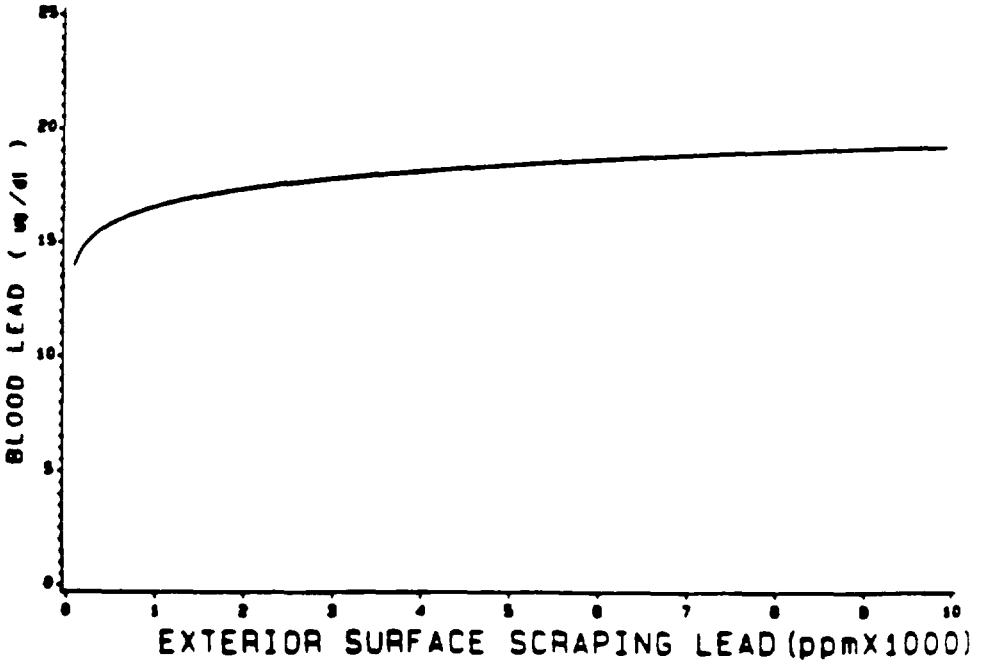


FIGURE 4- PREDICTED LEAD CONCENTRATION IN BLOOD (PbB) FOR GIVEN LEAD CONCENTRATIONS IN PbSS. THIS RELATIONSHIP REFLECTS ADJUSTMENTS (CONTROL) FOR XRFHAZ, PbD and PbH. THE REDUCED (ADJUSTED) STRUCTURAL EQUATION IS: ln (PbB)=2.345 + .067 (PbSS).

and seasonal factors as well as adjustment for previous Pb exposure history and developmental changes in the nature of the child's interaction with his or her environment.

CONCLUSION

The results of these interim analyses of the Cincinnati cohort suggest the following a) Lead contamination of the inner city residential environment is high and quite variable both within and between various housing categories. b) There appears to be a dust lead gradient in most homes with highest levels outside, next highest levels at the entrances to the dwelling and lowest levels within the dwelling. c) There is evidence to support the hypothesis that exterior environmental lead can result in blood lead elevations through the path PbSS -->PbD-->PbH-->PbB. d) An increase in PbSS from 0 to 1000 ppm results in an indirectly mediated increase in PbB of 6.2 ug/dl.

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For 18 month olds:

Reduced equation for the impact of PbSS in the presence of varying degrees of Paint Hazard.

$$\ln PbB = 2.297 + 0.081 \ln(XRF\ Hazard) + 0.067 \ln(PbSS)$$

18 month cohort $\bar{x}$ XRF Hazard = 1.74

18 month cohort $\bar{x}$ PbSS = 1360

$$\ln PbB = 2.297 + .081 \ln(1.74) + .067 \ln(1360)$$
$$\ln PbB = 2.297 + .045 + .48$$
$$\ln PbB_{18} = 2.822$$
$$PbB_{18} = 16.8\ ug/dl$$